MEASUREMENT OF CEREBRAL RESERVE CAPACITY USING ACETAZOLAMIDE LOADING XENON CT CBF BEFORE CAROTID ENDARTERECTOMY

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SUMMARY

Background and purpose: Brain swelling and/or hemorrhage can occur after carotid endarterectomy. This phenomenon is called the hyperperfusion syndrome. Several factors contribute to this syndrome. One is reperfusion in a maximally dilated vessel which means disappearance of cerebral reserve capacity (CRC). The aim of the study was to determine whether CRC measurement was useful for intraoperative and postoperative management of carotid endarterectomy.

Patients and methods: We studied 64 cases (male 53, female 11), 49-79 years. CRC was measured preoperatively using acetazolamide loading Xenon CT CBF examination (XeCT). Hypothermia (34-35°C) was induced during surgery in a patient with no CRC. Anesthesia was maintained the night after surgery and the systolic blood pressure was controlled below 120mmHg.

Results: CRC was absent in 10 patients. Postoperative CT did not reveal any hemorrhage or brain swelling. One patient experienced a transient restless state.

Discussion and conclusions: Cerebral hyperperfusion syndrome has been reported in 0.3~6.0% of patients following carotid endarterectomy (vs 1.6% in our study without hemorrhage or brain swelling). These data suggest that information on CRC could be useful for selection and perioperative management of patients during carotid endarterectomy.

Key words: hyperperfusion, endarterectomy, cerebral reserve capacity, acetazolamide Xenon CT.

INTRODUCTION

Brain swelling and/or hemorrhage can occur after carotid endarterectomy (CEA). Several factors may contribute to this phenomenon called the hyperperfusion syndrome. One of them is reperfusion in a maximally dilated vessel indicating loss of cerebral reserve capacity (CRC).

The aim of the study was to determine whether CRC measurement was useful for intraoperative and postoperative management of CEA.

PATIENTS AND METHODS

We studied 64 patients with stenosis (≥70%) of unilateral internal carotid artery (ICA) during the past 5 years. They included 53 men and 11 women, with an age range of 49 to 79 years old. Xenon computed tomographic cerebral blood flow examination (Xe-CT) was performed within 3 weeks before carotid endarterectomy. CRC was assessed by Xe-CT with acetazolamide loading test.

We induced hypothermia (34-35°C) during operation in one patient with CRC below 5ml/100g/min. This 70 year old man suffered from transient motor weakness of left extremities. On admission, he had no neurological deficit.
Neuroradiographical examination revealed multiple infarctions with watershed zone infarction in the right cerebral hemisphere (figure 1). Digital subtraction angiography showed a severe stenosis of the right cervical internal carotid artery (figure 2). The right middle cerebral artery was filled through anterior communicating artery supply during digital compression of the right carotid artery (figure 2) but left hemiparesis and slowing of electroencephalographic waves occurred during digital compression of the right carotid artery. Xe-CT with acetazolamide loading test revealed a steal phenomenon in the right middle cerebral arterial region (figure 3). A carotid endarterectomy was performed under hypothermia (34~35°C) and using internal shunt and Sendai cocktail. Anesthesia was maintained the night after surgery using propofol (3~5mg/kg/hr). The systolic blood pressure was controlled below 120mmHg. Furthermore the patient remained in bed for three days after the anesthesia and his systolic blood pressure was maintained within the range of 120~130mmHg with nicardipine hydrochloride or diltiazem hydrochloride.

He woke up without neurological deficit the next morning. He had no symptom of hyperperfusion syndrome such as restless confusion or headache. Postoperative neuroradiological examinations revealed no bleeding, swelling or additional infarction. Disappearance of the right carotid stenosis was confirmed at postoperative MR angiography (figure 4). He was discharged with no deficit.

RESULTS

The cerebral blood flow reserve capacity was markedly decreased to less than 5ml/100g/min in 10 of 64 cases (figure 5). All patients were men. The age ranged from 49 to 79 years old with a mean of 69. This age range was the same as the age range of the group with adequate CRC.

Postoperative CT did not reveal any hemorrhage or brain swelling. MRI showed no additional infarct. Only one patient experienced a transient restless state. In this series, morbidity was 1.5% and mortality was 0%.
DISCUSSION

In the literature, cerebral hyperperfusion syndrome occurs 0.3\%–0.6\% after carotid endarterectomy [1, 5, 6]. Neuroimaging studies show unilateral hemispheric edema and intracranial hemorrhage (ICH) in some cases with the syndrome [2, 7]. ICH carries a high rate of mortality and morbidity. Henderson and colleague reported that 12 (0.4\%) of 2,747 patients who underwent CEAs suffered postoperative ICH [2]. Seven of the 12 patients with ICHs died. They speculated mechanisms of brain edema and ICH after CEA are as follows. Some patients with severe carotid stenosis have impairment of cerebral autoregulation. In these patients, CBF increases markedly following CEA. High intracapillary pressure in maximally dilated vessels disrupts capillary endothelial cells and destroys blood brain barrier resulting in edema formation. If anticoagulation or perioperative ischemic event add to this condition, cerebral hemorrhage occurs after CEA.

According to their idea, it is important that the state of autoregulation be evaluated preoperatively in when planning CEA. The state of autoregulation can be evaluated indirectly by measuring reserve capacity (RC). Cerebral RC can be assessed by single photon emission computed tomography (SPECT), positron emission tomography (PET) and transcranial Doppler (TCD) with acetazolamide loading test [3, 4, 9]. Sbarigia and colleague reported that 3 of 36 cases had a loss of RC on TCD with acetazolamide test and these 3 cases had postoperative headache [8]. Yoshimoto and colleague reported that preoperative rCBF was significantly lower in cases of hyperperfusion syndrome than the control cases [9]. Moreover RC was evidently lower in the hyperperfusion cases than the control. Hosoda and colleague reported that SPECT with acetazolamide loading could identify patients at risk for hyperperfusion [3].

In our study, cerebral RC was assessed by Xe-CT with acetazolamide loading test. We could detect loss or marked reduction of RC in 10 of 64 cases. Xe-CT can be repeated at short time intervals, and thus is suitable for the acetazolamide loading test. Xe-CT is advantageous over other methods because of its ability to quantitatively evaluate data.

Yoshimoto and colleague reported that in cases of marked low perfusion with poor RC, hyperperfusion after surgical revascularization can occur even if blood pressure is adequately controlled [9]. In their series, the hyperperfusion syndrome occurred in 3 (6.5\%) of 46 cases. In our series, the occurrence rate of hyperperfusion syndrome was 1 (1.6\%) of 64 cases. There was no hemorrhage or brain swelling. We managed patients with poor RC by hypothermia during surgery and overnight anesthesia after surgery. Intraoperative hypothermia could prevent destruction of BBB more effectively, and overnight anesthesia allows strict control of blood pressure.

Our multidisciplinary management is useful for preventing of hyperperfusion syndrome after CEA.

CONCLUSION

We speculate that information on cerebral blood flow reserve capacity is useful for management of carotid endarterectomy intraoperatively and postoperatively. Measurement of cerebral reserve capacity using acetazolamide loading Xenon CT CBF examination is necessary for the planning of carotid endarterectomy.

REFERENCES